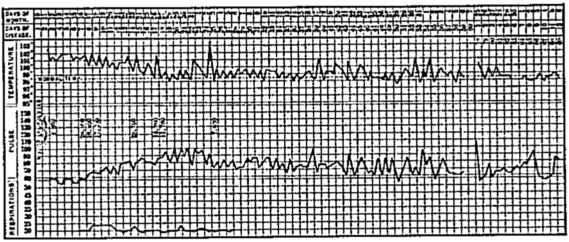
Urine negative, except for a very slight trace of albumin. On the seventh day there was a leucocytosis of 28,000 and an abundance of fibrin in the freshly examined blood. Still more diagnostic was the stiff neck which appeared now for the first time. This was so pronounced that the whole body was raised if the head was lifted. Motion of the head sideways was also impaired. On the eighth day there was no headache, the neck was less stiff, and the leucocytosis had dropped to 17,200. In agreement with this was the temperature, which was now running at a degree's lower level, namely, 100° to 101° F. For the next five days it gradually, but irregularly, fell, and the pulse rose until, on the fourteenth day of the disease, it again reached 102° F., the pulse 80, and the leucocytosis 28,700. The following day it was noted that a watch could be heard only within six inches of the ears. No mastoid tenderness. Hyperalgesia of the fingers and toes was quite pronounced. Kernig's sign absent. The knee-jerks were slight, and the abdominal reflexes were now obtained for the first time. With the exception of a high temperature on the twenty-second to the twenty-fourth day, the patient's condition steadily improved for three weeks. He ate well, was in good spirits, and had the merest trace of a headache. On the thirty-ninth day of his illness he vomited, and felt chilly and generally indisposed. His temperature rose once more to 101° F., and for two weeks it ranged between this point and the normal. Vomiting was repeated on the forty-second day, and again on the fifty-fourth, fifty-fifth, and fifty-seventh. Dr. E. M. Holmes kindly made an ocular examination on the fifty-eighth day, and reported normal, though rather pale, fundi.

CHART I.



As the patient had not improved for several weeks, he was moved into the country, where for sixteen days he gained steadily. During this time there was, with design, no record of pulse and temperature. He was seen daily, however, and it was thought that they rose but seldom above normal. About the seventy-fifth day he began to show less interest in what was taking place about him and said little. His appetite fell off and he vomited. Instead of going out-of-doors he kept his room. On the seventy-seventh day he was brighter, talked a good deal, and took a good amount of nourishment. The morning of the seventy-eighth day found him as usual. He did have a "little time" vomiting, but this seemed transitory. Toward noon, however, he grew restless and thought he must pass water or have an evacuation, but when he got up little or nothing came, and he needed help to get back to bed. On one of these occasions he had a convulsion, during which he was partially conscious, but made incoherent remarks. Two hours after this attack he presented the typical picture of a cerebral anæmia. He was conscious, but his pallor was extreme. The pupils were equally widely dilated and responded only partially and sluggishly to light. No paralysis of any portion of the body was detected. The pulse was 108, regular, of fair volume, but low tension. Three hours later the pulse had fallen to 80, consciousness was completely lost, and he again became very restless. At 8 P.M. the pulse had dropped to 56. He was barely conscious and responded with "um," "ah" to questions. Toward morning the restlessness grew less. Dr. Walton saw him on this day and demonstrated that he was aphasic. Examination, including that of the eyes, was otherwise entirely negative save for a doubtful right-sided facial paralysis. By the eighty-second day, four days after the convulsion, the aphasia had disappeared. For the remaining nine days of his life he lay quiet in bed, spoke only when addressed, and was confused about well-known facts. Occasionally he was restless, complained of headache, and would moan in his sleep. He rarely vomited. At times the urine and stools were passed involuntarily. Rectal temperature was, as a rule, 98° F., and the pulse was about 60 to 70 and regular. He took comparatively little food, and, though he retained nutrient enemata well, gradually failed. On the eighty-eighth day there was in the evening a suspicion of Cheyne-Stokes respiration. By the following morning it was plainly present. He continued to grow weaker during the day, and toward evening almost imperceptibly died. The autopsy was performed by Dr. Mallory, who diagnosed an internal hydrocephalus on hearing the history of the case. His report is as follows:

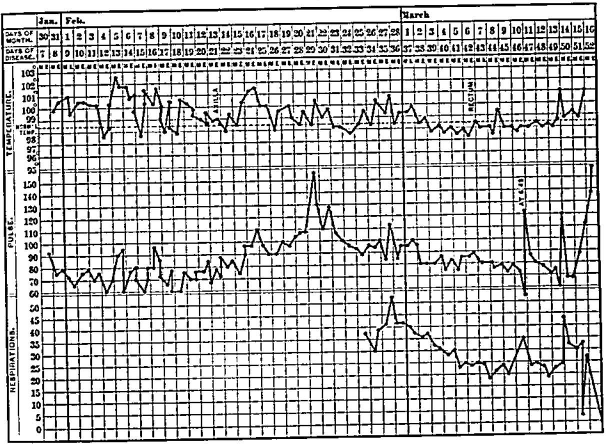
*Brain.* Dura tense; convolutions flattened; vessels of pia moderately injected; pia slightly thickened in places along the course of the large bloodvessels; thickening more marked along the fissures of Sylvius and at the base of the brain, especially around the pons and medulla and in the neighborhood of the foramen of Majendie. The floor of the third ventricle bulges. On separating the two halves of the cerebrum the corpus callosum rounds upward. On opening the lateral ventricles they are found considerably dilated and filled with a clear, watery fluid, estimated at 70 to 100 c.c. The third and fourth ventricles also show a little dilatation. The ependyma of all four ventricles, but especially that of the fourth, is swollen and velvety to the touch; the normal markings in the floor of the fourth ventricle are obscured. Further section of the brain showed nothing remarkable.

*Anatomical Diagnosis.* Internal hydrocephalus.

The case, then, in brief, is one of very mild cerebro-spinal meningitis which ran a fairly typical course for one month, with beginning convalescence; then came a month of stationary condition, during which there was slight pyrexia and an occasional attack of what was fairly typical cerebral vomiting. The first half of the third month showed steady improvement, which, however, terminated in threatening symptoms—apathy and vomiting—and these lasted three days; then came a convulsion with cerebral anæmia, from which the patient aroused slightly, only to pass gradually into an apathetic state, in which he succumbed on the eighty-ninth day of the disease. That the hydrocephalus had existed for some time; that an increase of the fluid took place about two weeks before the patient died seems probable, and that the pronounced cerebral anæmia, which occurred on the seventy-eighth day of the disease, was due to the closure of the foramen of Majendie—all these suppositions seem consistent with the events recorded.

CASE II.—Duration fifty-two days. For the following record I am indebted to Dr. Edward E. Hooker, of Arlington. G. T. F., aged forty-three years, during an epidemic of influenza, was seized on January 23, 1897, with severe headache, photophobia, and backache, which extended to the hips. On January 30th the patient was in a mild, muttering delirium, with hands picking at the bedclothes or holding the head as if in pain. Dejections involuntary during the next few days, and there was nausea and vomiting. On the twelfth day the temperature was subnormal, but it rose to 102.4° F. the next morning, when it was observed that there was slight twitching of the face and paralysis of the left levator palpebræ. On the sixteenth day the headache was worse, and the patient said his head felt swollen. Stupor, hiccough, nausea, vomiting, widely dilated pupils, partially closed eyes, and headache, together with involuntary dejections and a bladder requiring catheterization, constituted the picture on the nineteenth day. On the twenty-first day he began to moan, as if in agony, tossing and wringing his hands and rolling his eyes, and at times the thumb of his left hand turned in. The pulse rose to 140,' and the hands were

CHART II.



cyanotic. This condition was temporary, however. For the most part of the time until the thirty-fifth day he lay in a semi-conscious state, though with almost constant moving of the feet. The thirty-fifth to the forty-sixth days were variable. For some hours he was very bright, during others delirious. Respiration was deep and occasionally sighing. Gradually the daily amount of food taken grew less; temperature and pulse fell from 100.2° to 97.8° F. and from 110 to 55 respectively, and the respirations, which were 55 on the evening of February 27th, dropped to 20 on the morning of March 9th. The forty-sixth day found him too dull to swallow properly. The pulse varied between 55 and 120 in three-quarters of an hour, and in a slightly longer interval of time the respirations fell from 40 to 24. From now on he steadily failed; no new symptoms appeared, but there was an intensification of the old. For the last few days of his illness there were marked irregularities in the cardiac and circulatory rhythm, which the chart brings out. On the day before his death Cheyne-Stokes phenomena appeared. On March 16th he died.

*Autopsy* by Dr. McGrath. Cranium: Sinuses normal; dura mater normal except for slight adhesion between it and the pia at the base of the brain. Brain: Weight, 1500 grammes. Removal necessitates separation of the slight adhesions above noted; the pia mater appears somewhat thickened over the base and upon the upper and lower surfaces of the cerebellum. Otherwise external aspect not remarkable. On section both lateral ventricles are distended with clear, colorless fluid, in amount about 150 c.c.; the ependyma seems somewhat thickened. On dissection the pia in the vicinity of the foramen of Majendie; is notably thickened. Otherwise the brain is not remarkable.

Case III.—Duration ninety to one hundred days. M. C., laborer, aged thirty-two years, single, entered the service of Dr. George G. Sears at the Boston City Hospital on October 18, 1899. Previous history not obtained; had worked until about August 1st, when one hot day he had a fainting spell. At home a week with headache and lassitude; then he was able to walk about, but a week later began to grow worse. Occasionally delirious, and the headache at times was extreme. For two weeks he was somewhat deaf. Though in bed a month, his appetite remains good and the bowels are "all right." Two weeks ago he vomited. Examination: Well developed and nourished. Left pupil reacts slightly to light, right does not; pulse 76, regular, fair volume and strength; heart and lungs negative; abdomen somewhat retracted and tense, but not tender; liver and spleen normal; knee-jerks and plantar reflexes present. Grasp of the right hand weaker than that of the left, and there is a corresponding diminution in power of the right leg, with ataxia on standing. Urine contains no albumin. October 21st, good appetite and sleeps well; no pain; leucocytes, 19,000. October 23d, Dr. E. E. Jack reported a violent neuritis (choked), with hemorrhages and patches of degeneration. October 29th, feels very weak; no pain; eats well. November 3d, leucocytes, 6900. November 4th, feels "awful sick;" much headache; otherwise no change in his condition until death, on the following day.

*Autopsy* by Dr. Pratt. Head: Calvarium thin, especially the left frontal and parietal bones, which are in places not over 2 mm. thick; the dura is not adherent, but is very tense; the gyri depressed and flattened out; sulci effaced. The pia-arachnoid space over the cortex contains little or no fluid. The pia is somewhat opaque, and there are lines and collections of opaque white material along the vessels. This is apparently the remains of a purulent exudate, which has been replaced by connective tissue. The pia-arachnoid at the base is markedly thickened, and the pons, medulla, and cerebellum are bound together by a delicate network of fibrous tissue. Cover-slip preparation from the opaque material upon the cortex shows neither bacteria nor pus-cells. The interpeduncular space is so distended with fluid that it forms a distinct spherical cyst, projecting about 2 cm. above the surface of the brain when placed with the cortical side to the table. Lateral ventricles greatly distended with clear, pale fluid, 80 c.c. in amount. When the fluid escaped the cyst at the base collapsed. The fluid had a specific gravity of 1010, and contains one-twentieth of 1 per cent. of albumin. The foramina of Monro readily admit the little finger. The ependyma is smooth; the underlying cerebral substance is not softened; the choroid plexus is not swollen and is natural looking. The third ventricle and the aqueduct of Sylvius are markedly enlarged. The basal ganglia, particularly the corpora quadrigemina, are compressed. In the superior fovea of the fourth ventricle is a small, firm, yellowish-white opaque mass, several millimetres in size, that completely occludes the ventricle at this point. The inferior fovea is covered by a soft, reddish substance, apparently young connective tissue, that closes the foramen of Majendie and the apertures of Mierjejewski. Culture from the fourth ventricle sterile. Brain: Weight, 1340 grammes. The right middle ear is filled with thick, tenacious pus. Cover-slip preparation shows many polymorphonuclear leucocytes and numerous diplococci that stain by Gram's method. No other bacteria seen. Tympanic membrane and ossicles intact.

*Anatomical Diagnosis.* Chronic leptomeningitis. Chronic internal hydrocephalus, secondary to the occlusion of the fourth ventricle. Acute suppuration of the right middle ear. Atrophy of the bones of the skull.

CASE IV.—J. E., aged fifty-eight years, married, born in Germany, and worked in a slaughter-house. On May 13, 1897, entered the service of Dr. Bowditch in the Boston City Hospital. Excessive beer drinker; "rheumatism" for nearly a year, and "sleeping spells." Two days before entrance drowsiness and stupor appeared, with pain in both ears, but without headache. On the day of admission, nausea. Examination: Well developed and nourished; semi-conscious, at times delirious; slight ptosis of the left eye; left pupil smaller than the right; neither react. The tongue protruded evenly; considerable tenderness exists below the mastoids. Pulse regular, of good strength and volume; arteries thickened and tortuous. Chest and abdomen negative; knee-jerk on the right, but left diminished. Cremasteric reflexes sluggish; urine normal; temperature 99.4° to 96.2° F.; pulse 65 to 90, and respirations 17 to 28. May 16th, the patient, at times delirious, but generally stupid, became unconscious and died May 19th.

*Autopsy* by Dr. Mallory. Brain: Dura about normal in thickness; pia much thickened over the whole brain, white and opaque; thickest and whitest along the large bloodvessels. It corresponds exactly with an acute inflammatory exudation in the meninges, except that it is white and opaque instead of yellowish. In its thickest portions the pia was exceedingly tough and could with difficulty be torn.

It stripped off from the brain-substance rather easily, and did not tear it much. Over the lateral portions of the brain the exudation was less marked, confined almost wholly to the neighborhood of the larger vessels. In places at the apex it was thick enough to obscure the brain tissue and the vessels beneath. In its meshes was a considerable amount of serum. At the base of the brain was an abundant whitish exudation, most marked over the medullary and upper portions of the cervical cord. The brain was removed with difficulty on account of its being necessary to cut through the adhesions between this portion of the cord and the foramen magnum. The optic nerves were swollen and soft and the commissure was shoved inward. Just back of it, surrounding the pedicle of the pituitary body, was a firm, grayish, translucent mass. The cranial nerves, other than the optics, seemed to be normal. There was little exudation over the pons or along the fissure of Sylvius. On opening the brain the lateral ventricles were found to be considerably dilated, containing each 30 to 40 c.c. of fluid. The ependyma was slightly granular. The bloodvessels were moderately injected. In the third ventricle was a small mass of whitish fibrin, attached at one end of the wall. In the fourth ventricle was a mass measuring 1.5 cm. in diameter and 3 mm. in thickness, unattached to the wall; its edges were rounded and extremely like a small piece of decolorized fibrin. The sides of the corpus striatum adjoining the third ventricle were swollen, firm, and transparent, and resembled closely the tissue described as lying just back of the optic commissure; on each side it was about 1.5 cm. in width. Outside of this on both sides, but particularly on the left, the central portion of the corpus striatum was very œdematous, soft, and transparent.

*Anatomical Diagnosis.* Organization of acute cerebro-spinal meningitis, with occlusion of the foramen of Majendie and the production of internal hydrocephalus.

CASE V.—Duration sixty days. A. A. D., aged sixteen years, single, a brass and iron filer, entered the service of Dr. Vincent Y. Bowditch at the Boston City Hospital on May 2, 1898. As a child he had measles, and at four years scarlet fever, followed by "kidney trouble." Habits good. About the last of March he had a fight, was hit on the mouth and knocked backward against a wall, striking his head and neck. Present illness began two weeks before entrance, with languor and insomnia. While he was having these symptoms a man, with whom he lived up to February 2d, came to the hospital and died with cerebrospinal meningitis. Three days before entrance he was suddenly seized with violent frontal headache, with dimness of vision. Worked until 6 P.M., when he vomited several times. The next day headache was more severe, and he had several chills and vomited. Dimness of vision increased; he staggered in walking, with a tendency to fall to the right. Toward night he became delirious. On the day before entrance delirium alternated with rational spells. Fell out of bed. A macular eruption appeared on the face, which disappeared within twenty-four hours. Pulse irregular and very slow. On the morning of entrance he had a convulsion; rigid, with hands clenched; cyanotic and stertorous respiration. Retraction of the neck was now for the first time noticed. Bowels constipated, but at no time involuntary defecation or urination. Examination: Well developed and nourished; stupid; flushed face; pain on pressure on the back of the neck, and slight pain on movement of the retracted head. Right pupil larger than the left; both react. Conjunctivæ congested, and there is a questionable external strabismus. The tongue is covered with a moist, brownish coat; sordes about the teeth. Heart is normal except for prolongation of the first sound at the apex; pulse normal. Lungs and abdomen negative except for enlarged spleen. Knee-jerks lively. Small petechiæ over the skin. Leucocytes, 20,600. Urine normal. May 4th, slight ptosis of the left eye. May 5th, leucocytes, 9000. May 7th, steady improvement; no pain. May 8th, Dr. Lancaster reports a slight swelling of the optic nerve, with the veins considerably distended and tortuous; a few minute hemorrhages in retina near the disk; beginning optic neuritis. May 16th, patient doing well; no new symptoms except diplopia for distant objects. On May 20th he was discharged "well." The temperature did not rise above 99.4° F., and the pulse was for the most part of the time about 70, but was in the 50s on three occasions and just before discharge. June 17th, re-entry in the service of Dr. Henry Jackson. The patient had been comparatively well since leaving the hospital save for general bodily weakness, and on this account has attempted no physical exertion. Yesterday he felt so well that he rode a bicycle, but came home tired and with intense frontal headache. Went to bed and slept. About five o'clock this morning he got out of bed, walked ten feet, and had a convulsion, falling unconscious to the floor. At nine o'clock he had a second convulsion, at one o'clock a third, and at four o'clock a fourth, lasting about eight minutes, with frothing at the mouth. At 4.30 P.M., during another convulsion, he died. Examination made shortly before death showed him to be semi-conscious, delirious, opposed handling. Little, if any, stiffness of the neck; motions of the head resisted. Marked left external strabismus, with moderately dilated pupils, which react sluggishly to light. Pulse regular, full strength and volume. Heart, lungs, liver, and spleen negative. Knee-jerks lively. Leucocytes, 26,160. Temperature, 99° F.; pulse, 108; respiration, 29.

*Autopsy.* Brain weighed before opening 1650 grammes; after opening, 1530 grammes; 120 grammes of fluid. Dura tense. On stripping it back the surface of the brain was moderately injected; very dry. No thickening of the pia can be made out anywhere, unless possibly a little around the optic commissure and posterior to it. The floor of the third ventricle projects outward 3 mm. On opening the brain, the lateral ventricles are found to be distended and filled with a perfectly clear, watery fluid. No evidence of any inflammatory exudation can be found in the posterior cornua. The ependyma shows very slight granulation. The third ventricle contains a spherical, soft, yellowish cyst, measuring 13 mm. in diameter. It is adherent at several points to the choroid plexus of the third ventricle. Its wall is yellowish and to some extent translucent. On section the tumor is found to be encysted with a thin, delicate wall; the contents are grayish, rather opaque, and tenacious, sticking more or less together. Microscopical examination of this material shows it to consist partly of leucocytes, partly of large cells filled with fat-drops. The tumor mass obstructs the iter a tertio ad quartum. The wall of the third ventricle is thickened and granular. At the tip of the temporal lobe on the right side is a small, yellowish area, 2 mm. in diameter. Cyst due to puriform softening of the mass of fibrin in the third ventricle following cerebro-spinal meningitis. Obstruction of the iter a tertio ad quartum. Internal hydrocephalus.

Case VI. is not as typical as the others, but will serve as the connecting link between the acute cases and the chronic ones, in which the hydrocephalus fully developed. Duration forty days. R. P., an errand girl, aged seventeen years, born in Germany, entered the Boston City Hospital on October 21, 1898, in the service of Dr. George B. Shattuck. Family history negative. Usual children's diseases and diphtheria. Present illness began three weeks ago, with pain in the right arm, extending to the shoulder and down the back to the lumbar region. Intense pain in the frontal and apical region of the head, also pain and tenderness in the back of the neck. Considerable photophobia. For the first three days of sickness there were numbness and "pins and needles" in the legs and inability to move them. Chills nearly every night. In the chest is a feeling as of suppressed hiccoughs, with a slight hacking cough. Tonsils somewhat enlarged and painful when yawning. Micturition was accompanied by a burning, cutting sensation. Yesterday a temporary aphasic attack, lasting for ten minutes, and to-day another for five minutes. Vomited frequently at first, later irregularly. Examination: Well developed and nourished. Temperature, 100° F.; pulse, 90; respirations, 10. Pupils equal, dilated, and react. Neck painful to pressure. Heart and lungs normal. Spleen slightly enlarged. Reflexes normal. Leucocytes, 19,000. Widal reaction negative. On the day after entrance the patient had a severe chill. Mentality unimpaired, but in the afternoon a temporary paralysis of the right side of the face. October 24th, another chill, and headache so intense that she shrieked. October 26th, eyes and ears drawn to the left. Some difficulty in following the movement of the finger beyond the median line to the right. Constant twitching. Increased left patellar reflex and an ankle-clonus persisting for many minutes. A loud systolic murmur at the apex and over the whole cardiac area, which later disappeared. October 27th, clonus absent. October 31st, semi-conscious. November 4th, unconscious; temperature moderate; no more chills. November 8th, for the past two days temperature, pulse, and respiration all elevated; semi-retraction of the head; labored breathing. November 9th, died.

*Autopsy* by Dr. Mallory and Dr. Defendorf. Brain weighed 1295 grammes; dura rather tense. On removing it convolutions are a little flattened; vessels of the pia injected a little more than normal over the vertex of the brain; no thickening of the pia arachnoid; the olfactory bulbs are adherent to the skull. On removing the brain quite a little fluid escapes from around the base. On the under surface of the brain, over the circle of Willis and slightly involving the optic commissure, there is a whitish, rather translucent, tough deposit, varying from 2 to 3 mm. in thickness. A platinum needle could be drawn through it with difficulty. A little of a similar material is present over the vermiform process of the cerebellum and extending out toward the fissure of Sylvius. Otherwise than for this material the surface of the brain presents nothing abnormal. On opening the brain the lateral ventricles are somewhat distended with fluid, at least 20 c.c. each. The ependyma is everywhere finely granular. Over the corpus striatum the granules are decidedly prominent and larger than one ordinarily sees in this condition. In the right ventricle and in the inner posterior corner of the corpus striatum, just where it joins the optic thalamus, is a small, yellowish, opaque elevation, 4 mm. in diameter and projecting 2 mm. above the ependymal surface. In the posterior cornua of both lateral ventricles is a soft mass of yellowish material, forming a little over 1 c.c. in each horn. Microscopically this contains more or less broken-down fibrin and a few pus-cells. The third ventricle is a little dilated and its ependymal surface decidedly granular. A similar condition is present in the fourth ventricle. In the lateral pockets is a little yellowish material similar to that in the horns of the lateral ventricle. The substance of the brain presents nothing abnormal microscopically.

*Anatomical Diagnosis.* Organizing meningitis. Granular ependymitis. Moderate degree of internal hydrocephalus.

CASE VII.—Duration eighty-two days. Maggie D., aged twenty-four years, entered the service of Dr. W. W. Gannett at the Massachusetts General Hospital on October 2, 1897. Family history good. Two weeks before entrance she buried a child with cerebro-spinal meningitis. Four days before entrance she vomited without cause, and on the following day was attacked with headache, backache, and vomiting, which again was of sudden onset, and, in conjunction with diarrhœa, caused great weakness. Examination: Well developed and nourished; good color, many herpetic vesicles about the lips. Pupils reacted, but were unequal, which the patient said was always the case. Neck rigid and tender. Chest and abdomen normal. Knee-jerks and plantar reflexes present. Leucocytes, 23,600. Diplococcus intracellularis Weichselbaum was grown from the nasal discharge. Vomiting and headache continued after entrance. The pulse was irregular, and fresh crops of herpes appeared. Between the seventh and twelfth days she improved, but occasionally had diplopia. Many hyperæmic papules appeared on the thighs and legs, which later changed to pustules. During the next week she was better and worse by turns. At times the heart was weak and the respiration suggestive of the Cheyne-Stokes type. Leucocytes on the nineteenth day, 10,600. On the twenty-fifth day the eyes were examined by Dr. Cheney: Slight congestion of the retinal veins. Persistent vomiting, with resulting emaciation. Leucocytes on the forty-eighth day, 9600; on the fifty-fourth day, 8200; on the sixtieth day, 7000; and on the seventieth day, 16,200. The left pupil lost its reaction

CHART III.

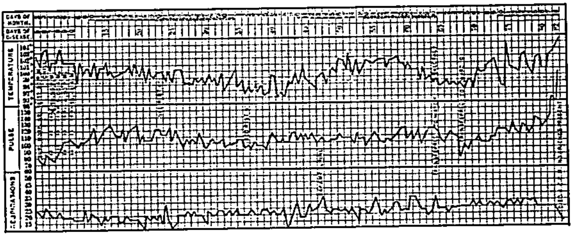
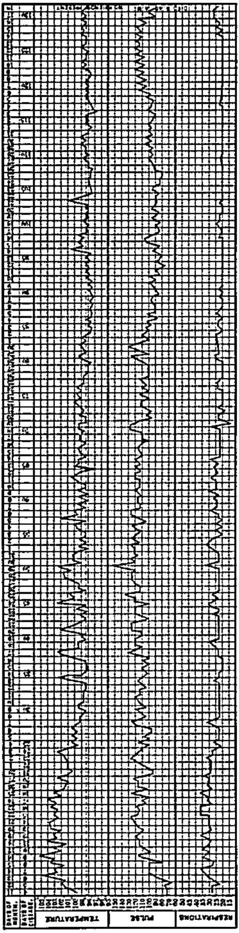


CHART IV.



to light on the eighty-first day, which was the day of her death. For some days previous the knee-jerks had not been obtained. Vomiting, however, ceased during the last week of life. Cheyne-Stokes respiration at times manifest.

*Autopsy.* Meninges of convexity dry, not specially injected, and showing no exudate. Convolutions somewhat flattened. At the base over the pons and a portion of the medulla the meninges were infiltrated, with a yellowish, semi-translucent, somewhat jelly-like material. The region of the chiasma was free from exudate. On removing the brain a stream of clear, watery, colorless fluid squirted from an opening in the neighborhood of the optic chiasma. Lateral ventricles and fourth ventricle markedly dilated, and contain a rather cloudy fluid. In the lateral ventricles some large, fibrinous flocculi were present. Dissection of the brain shows nothing remarkable except that the tissue of the corpus callosum and fornix posteriorly is perhaps softened. Lateral sinuses free. Cord: No accumulation of fluid in the subdural space. In the cauda equina and in the meshes of the pia of the thoracic and lumbar portions are accumulations of opaque, yellow exudate arranged in patches. Exudation is not extensive. In the thoracic portion the cord seems softer than normal; this may be due to injury in removal.

*Anatomical Diagnosis.* Subacute cerebro-spinal meningitis; internal hydrocephalus.

CASE VIII.—Duration one hundred and thirty-one days. (Chart IV.) J. C. entered the service of Dr. R. H. Fitz at the Massachusetts General Hospital on February 11, 1898. Parents and one sister died of phthisis, and another sister has the disease. Except for bronchitis at three years of age he has been well. February 8th he was exposed to cold and wet, and felt chilly, had pains in his arms during the night, and was delirious.

February 9th, headache and vomiting commenced and persisted. Examination: Fairly developed and nourished man, in much pain. Pupils small; reaction sluggish. Neck and back stiff. At the apex and base of the heart a faint systolic murmur. Knee-jerks present. Leucocytes, 36,000. Urine slight, trace of albumin, rarely a hyaline cast; no diazo reaction. During the next few days delirious and in pain. Leucocytes, 12,700 on the sixth day, but 20,000 on the eighth. On the following day he developed a pneumonia of the right lower lobe, with a leucocytosis of 34,000. Four days later, still very ill, and leucocytes 40,300. On the seventeenth day a patch of pneumonia appeared in. the left back, and the white corpuscles were 50,800. For the next seventeen days he continued to improve, but the white count fell only to 26,000. By the fifty-ninth day a living skeleton; answers "yes" and "no" and "fine;" lies with his eyes closed and arms folded Micturition involuntary and vomiting frequent. On the twenty-fifth day he had a chill, and leucocytes 45,700. No plasmodia. A bacillus similar to the tubercle bacillus was found in the sputum, but pronounced by Drs. Ernst and Wright not to be the tubercle bacillus. From about the sixtieth day on the temperature was usually normal or subnormal. At times there appeared to be some slight improvement in his condition, but on the one hundred and sixth day his weight was 63 pounds instead of 130 pounds as at entrance. Leucocytes still increased, being 26,000. On the one hundred and twenty-second day, failing slowly and more emaciated. Skin dark; bloodvessels distended; bedsores on the back, thighs, legs, knees, ankles, elbows, and shoulders, and there seemed to be only just enough tissue left to hold the bloodvessels together. Cheyne-Stokes respiration ushered in the end on the one hundred and thirty-first day.

*Autopsy.* Chronic leptomeningitis of the base of the brain and of the spinal cord, with hydrops ventriculorum and softening of the right lenticular nucleus. Head: Dura normal; pia of convexity normal; convolutions flattened. At the base an accumulation of thin, cloudy fluid. The pia at the base shows fibrous shreds and some thickening. Frontal lobes more adherent than normal. Lateral ventricles considerably dilated. A large part of the right lenticular nucleus mushy, soft, and brown in color. Vessels and cerebral tissue otherwise not remarkable. Middle ears normal. Spinal cord: Dura everywhere rather firmly adherent to the pia-arachnoid by translucent tissue. Cervical cord fluctuating to the touch. On section of the dorsal and lumbar cord the nerve tissue seems to show extensive softening, is soft and mushy. No fluid accumulation in the dural sac. The cervical region seems somewhat enlarged and fluctuant.

### *A Résumé of the Literature on Internal Hpdrocephalus following Cerebro-spinal Meningitis.*

Internal hydrocephalus has been known to be a result of epidemic cerebro-spinal meningitis since 1805, the year in which the disease is usually considered first to have appeared.

In the same year[1] (1805) a case occurred at Strasburg, terminating fatally after two relapses, the last one being accompanied by cerebral symptoms. At the post-mortem the vessels of the head and membranes were engorged with blood; serous fluid was found between the meninges above the hemispheres. The brain was soft, the ventricles were dilated and contained about four ounces of turbid serum, with a deposit of pultaceous, puriform matter lining their cavity. (*Journ. de Med., Chir. et Phar.*, etc., vol. x.)

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[1] Quoted by Webber. Boston Medical and Surgical Journal, vol. lxxv., p. 85.

Foerster,[1] in 1863, gave a very good description of this complication. He says the inflammation usually runs a fatal course, a recovery being seldom complete; sometimes the acute symptoms cease and the case becomes chronic. Meanwhile the secretion of serum continues and forms a chronic hydrocephalus. The ependyma at times remains forever generally or partially thickened.

Ziemssen and Hess[2] published a long article upon "Clinical Observations in Epidemic Cerebro-spinal Meningitis," in which they record four cases of long duration which showed at the autopsy internal hydrocephalus. No decision was reached as to whether the process was inflammatory or due to a passive congestion from pressure on the veins of the choroid plexus.

To this excellent account Ziemssen,[3] in 1874, added a more extensive description, which to-day embodies more than any other article on the subject. He says that lasting alterations in the brain, the spinal cord, and their membranes are not by any means seldom. Among these chronic hydrocephalus appears to form far and away the most frequent after disease. He explains this change as follows: The slightly altered, perhaps thicker, exudation is absorbed slowly or quickly in the second week, through fatty degeneration of the cells and fibrin, or, in consequence of absorption not occurring, shrivels up into a caseous mass. While the connective tissue of the soft membranes proliferates the hyperæmia of the brain-substance subsides and the purulent effusion in the ventricles increases. In the twenty-seventh or twenty-eighth week the soft membranes show a pulpy hyperplasia or already cicatricial thickening; the caseous remains of the exudate contract still further; the effusions in the ventricles become more moderate in amount, but quite clear, while the cellular elements in the dependent parts of the ventricles are thickened to small, caseous plates. The earlier hyperæmia of the brain is completely gone; the brain is anæmic, even œdematous; the ependyma of the ventricle is thickened and clearly granulated, and the choroid plexuses bloodless. The diminution of the brain substance depends on the degree of the hydrocephalus, but can be very considerable. It almost appears as if the increase of the ventricular effusion was conditioned in the first instance by the contractions and thickenings of the soft membranes. At least the usual stage of apparently progressive convalescence between the acute attack of meningitis and the appearance of the hydrocephalic signs speaks for this view.

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[1] Path. Anat., 1863, p. 597.

[2] Deutsches Archiv., 1865, vol. i.

[3] Handbuch d. acuten Infect. krankheit., Zweiter Theil, 1874, p. 683.

The symptoms of the secondary hydrocephalus consist of paroxysms of severe headache, and also of pains in the neck and extremities, with vomiting, unconsciousness, convulsions, involuntary evacuations of feces and urine.

These attacks recur either in a pretty regular type or make pauses which not rarely last for weeks, and during which the activity of the mind and senses may be unimpaired, the appetite excellent, the sleep restful, and the patient in good spirits and gaining strength. If these interims last for a considerable time we easily fall into the error of supposing that the disease has taken a favorable turn, until a new attack dispels the illusion. Generally, however, the condition during the interval is not so favorable that such a prognostic error is possible; there persists the general hyperæsthesia, increased reflexes, furthermore slight contractures or pareses of the extremities, psychical disturbances, and other symptoms.

Of great practical importance is the question whether and to what degree these hydrocephalic effusions are capable of retrogressive changes; in other words, is cure of a secondary hydrocephalus ever possible? Generally it is not; "yet I have seen some cases in which a complete, and others in which an incomplete, recovery took place."[1]

In the interval between Ziemssen's two publications Collins[2] gave an excellent report upon "Epidemic Cerebro-spinal Meningitis." He says a return of this symptom (pain in the head) in company with vomiting and convulsions but too frequently points to the issue of the disease in hydrocephalus. He records the case of a boy, aged ten years, who died on the sixty-sixth day of his illness, with, he thinks, the cause of death due to an occlusion of the cerebro-spinal opening. This is the first direct mention of this as the cause of the hydrocephalus in such a case that I have been able to find.

In 1876, Neville Hart[3] recorded four or five typical cases of hydrocephalus following meningitis, and in describing the symptoms it is interesting to note that dilatation of the pupils was constant in the late stages. The respiration was irregular, there was great emaciation, bedsores, paralysis of the bladder and rectum, and in a few cases of the pharynx.

Heubner[4] describes this type of cerebro-spinal meningitis. He says that hydrocephalus is to be recognized by paroxysms of pain, vomiting, coma, and convulsions. The weakness of memory, intelligence, and speech may be due to the same cause.

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[1] Archiv f. klin. Med., Band l., p. 389.

[2] Dublin Quarterly Journal, 1868, vol. xlvi., p. 170.

[3] St. Bartholomew's Hospital Reports.

[4] Eulenberg's Encyclopædia, third edition, pp. 430, 435, 437.

Eichhorst[1] says he has repeatedly seen hydrocephalus develop, and in two cases far advanced in convalescence there suddenly appeared headache and apathy, which resulted in a few hours in death, for which the only cause was a marked internal hydrocephalus.

Von Leyden[2] calls attention to epileptic convulsions as characteristic of meningitis of the convexity, but to coma and cerebral paralysis as significant of large exudations in the ventricles. Later,[3] he says the danger of the disease lies more in the danger to the brain-substance through the exudate than through the severity of the infection.

This by no means exhausts the literature, but most of the articles written on the subject are quoted under one or another of the above references or are referred to on page 128 of the *Report of the State Board of Health of Massachusetts*, 1898, which was written by Councilman, Mallory, and Wright.

PATHOLOGY. A comparison of the reports in our eight cases of cerebro-spinal meningitis shows a marked uniformity. In all save one the hydrocephalic fluid was clear and colorless, and was sterile in two instances in which it was examined; in a third, diplococci were found, though not the specific diplococci of the disease under discussion. (The presence of bacteria in this case (VII.) may account for the peculiarity of the high temperature which existed in the latter weeks of the illness and rendered the case unique in this respect—*i. e.*, a secondary infection may have taken place. This is by no means certain, however, as the bacteria were few in number.)

That the hydrocephalic fluid was sterile we should expect, for it is an acknowledged fact that the bacteria in these cases disappear in the early part of the disease. Akin to this is the absence of collections of pus, save in the cases of shortest duration (VI. and VII.), in which leucocytes were found in the cornua of both lateral ventricles.

The pia is reported thickened in all cases in the region of the roof of the fourth ventricle, and in most of the cases elsewhere as well, particularly at the base of the brain. The thickening was of marked degree, in one case resisting the thrust of a platinum needle, in another necessitating a division of the adhesions. This thickening is due to organization—that is, to the formation of granulation tissue, which starts from the pia and grows into the fibrinous exudation, gradually dissolving and removing it. It covers enough surface not only to bar the foramen of Majendie and the two lateral openings of Mierjejewski, but to render impervious the whole roof of the ventricle.

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[1] Hand. der spec. Path. u. Therapie, 1897, p. 527.

[2] Zeitschrift für klin. Med., 1887.

[3] Nothnagel Series, vol. x., p. 291.

There does not appear to have been any thickening of note in the above cases about the bloodvessels except in Cases III. and IV., nor were the choroid plexuses injured.

The changes in the brain resulting from the increased pressure were not different from those of other cases of hydrocephalus. The flattening of the convolutions varied with the amount of fluid. The bloodvessels of the pia were, as a rule, injected.

There is less uniformity in the condition of the ependyma. In the first case it was swollen and velvety, in the second somewhat thickened, smooth in the third, and very slightly granular in the fourth, fifth, and sixth. Nothing abnormal was observed in the seventh and eighth.

In the light of the records of the preceding cases and the observations collected from other sources, a very definite description of this sequel of meningitis can be given.

Unless the disease is rapidly fatal, toward the end of the second week the intense hyperæmia of the soft membranes subsides, and the fibrinopurulent exudate begins to undergo absorption in the same manner as in any other portion of the body. At the same time the large or small effusion in the ventricles, which is turbid with flakes of pus in the extremities of the cornua, changes its character. This purulent material is either absorbed, owing to the abundant vascular supply furnished by the choroid plexuses, or shrinks into caseous masses which are deposited on the walls of the ventricle. In either event, by the end of the third week, if not before, the fluid in the ventricles and subarachnoid spaces is clear, and, as experience has recently shown, is sterile. This latter fact is of especial importance, because it helps to exclude the idea of a chronic inflammation of the ependyma as a cause of this hydrocephalus.

During the next few weeks the organization of the exudate occurs, and in our opinion, according to the extent of this process of repair, which in turn is necessarily dependent on the location and amount of the exudate at the commencement of the disease, rests the final outcome of the case. If the nerve-roots have been embedded in the exudate they are liable to suffer from the cicatricial thickening about them. If the inflammation has been extensive and the meninges of the base of the brain "plastered" in the region of the roof of the fourth ventricle, the foramen of Majendie and the neighboring foramina of Mierjejewski may be obliterated and the velum interpositum thus rendered impervious. We have taken for granted that there is such a foramen and such neighboring foramina, as this appears to be the prevailing opinion; but should one not accept this supposition it would not alter our argument, for the whole velum is often covered with the exudate, and the whole might be easily rendered impervious. A hydrocephalus would then be the natural result.

In 1863, Hilton, in his *Lectures on Rest and Pain*, emphasized this factor in the etiology of hydrocephalus. He mentions and delineates the specimen from a case which he autopsied in 1844. Collins followed Hilton's lead, and in a case of cerebro-spinal meningitis of his own attributes the resulting hydrocephalus to the closure of the foramen.

But by no means is this the only method for the production of an intraventricular effusion non-inflammatory in character. In many cases the lateral and third ventricles are dilated, but the fourth not. In Case V. of our series this occurred, and the cause was manifest — there was an obstruction of the iter a tertio ad quartum. This is not, however, a unique occurrence, and it is only to be wondered at that this does not occur more frequently, for it is so easy for a collection of fibrin to become organized at this spot. Similarly the foramina of Monro may be plugged, one or both, and such would seem to be the case in those ordinary forms of chronic hydrocephalus in children in which puncture of one ventricle serves to drain only that side of the brain. Naturally there is no reason why the veins of the choroid plexuses should not be compressed by the cicatricial contractions; but it is not clear how this occurrence alone would explain the hydrocephalus, providing the paths for the circulation of the cerebro-spinal fluid were intact. It must be a complete obstruction of the veins, indeed, that would suffice to produce such an increase in the amount of cerebro-spinal fluid as to start in operation that vicious circle hydrocephalus leading to compression of the brain and its vessels, thereby preventing the removal of the excess of fluid and at the same time favoring an increase in the hydrocephalus.

SYMPTOMATOLOGY AND DIAGNOSIS. It is impossible to decide definitely when the hydrocephalus begins in these cases, because the symptoms of meningitis and hydrocephalus are in the first instance so similar, and in the second place both conditions may exist together from the start. In three cases (I., III., and V.) an abatement of symptoms or an interim of apparent convalescence followed the first attack, while in four patients (II., VI., VII., and VIII.) the active stage of the meningitis merged into a condition which was evidently directly attributable to the hydrocephalus. Case V., for example, so far recovered from his initial attack that he was able to take a bicycle ride, though with disastrous sequences. Case I., with some help, made his way up and down stairs. Case VII., however, was of eighty-one days' duration, but presented no symptoms of hydrocephalus in the last month other than the ravages upon the system of a long illness, which were not present in the first four weeks of the disease.

These cases further show that no relation exists between the development of the hydrocephalus and the severity of the original attack. Cases I., III., and probably IV., were of mild character, while in the others the disease was severe. From this we may infer that it is not so much the extent and intensity of the initial inflammation as it is the location of the same.

ANALYSIS OF SYMPTOMS. Apathy or mental lassitude on the part of the patients is the predominant factor in the symptomatology. The histories lead us to believe that this was present in each case which was under observation. Beginning with simply apparent drowsiness and indifference, it gradually increases until the patient appears asleep the greater part of the time. In reality he is not, for he is easily aroused and responds to a question, but only to drop off again into his former condition. One can trace the gradual increase of this lethargy in the steady diminution of voluntary efforts and remarks on the patient's part. He is profoundly passive. This state may pass into one of unconsciousness, as in Case II., and the patient remain in this condition until death. Usually this is temporary, and the patient finally dies somnolent rather than unconscious.

Vomiting took place in all but one of the eight cases. Its irregular occurrence, the absence of any relation to diet or treatment, and the lack of other gastro-intestinal symptoms stamped it as of nervous origin.

Headache was severe in one-half of the cases, but in three of the others it was distinctly of mild type, and in one patient was wholly absent. The somnolence of the patients may have masked its character, but it is certain that it was far less severe than the headache of onset.

The pupils were dilated or reacted sluggishly or not at all in seven of the patients. In the final case no note is made of their condition.

Muscular weakness and emaciation were the rule, but not out of great proportion to the length of the disease and the small diet.

The temperature curves are extremely suggestive, because they were of normal or subnormal character for much of the illness, especially for the few weeks before death. In Case II. there was a rise for about forty-eight hours before the end, and in Case VII. the curve is so similar to that of typhoid fever that we are glad to know its absence was confirmed by the autopsy. The relation of temperature to pulse is most striking. In Cases I., II., VI., and VII. the pulse repeatedly *rises* as the temperature falls, and *vice versa*. This reverse of the rule is not absolute, and holds chiefly for the early part of the disease. Toward the end the pulse falls, and then appear those great irregularities in rate from hour to hour which have always impressed the physician. These later irregularities, however, as well as the Cheyne-Stokes phenomenon, which occurred four times, are terminal manifestations.

Quite in accord with the pathological findings is the most striking clinical feature of these cases, namely, their long course. The duration of the various cases was successively eighty-nine, fifty-two, ninety to one hundred (?), sixty, forty, eighty-two, and one hundred and thirty-one days.

Mental apathy, vomiting, headache, dilated or sluggish pupils, with a normal or subnormal temperature when they occur a month or more after the onset of cerebro-spinal meningitis, point to hydrocephalus. Convulsions, paralyses, including aphasia, optic neuritis, dizziness, involuntary passage of excreta, and bed-sores may be concomitants, but they are less common and not as distinctive. With the absence of fever in these cases it is surprising that in Case III., five days before death, the leucocytosis was 19,000; in Case V., on the day of death, 26,000; in Case VIII., with an almost unbroken normal or subnormal temperature for weeks before and after, that it should be on the ninety-fifth day of the disease 26,000.

PROGNOSIS. Von Ziemssen[1] says he has seen some cases in which a complete, and others in which an incomplete, recovery took place. Hart[2] mentions two cases of recovery of the chronic form. Of course, in the nature of the case, positive data regarding recovery from the hydrocephalus are and must be uncertain, because of the difficulty of the diagnosis. In looking over the 111 cases recorded in the *Report of the State Board of Health of Massachusetts* I find that twenty-two cases were of forty or more days' duration. The mortality for the whole number of cases was 68 per cent., but of the twenty-two mentioned was 23 per cent. Of the seventeen cases which eventually recovered eleven had some definite signs of hydrocephalus, four evidently did not have hydrocephalus, while a mastoid abscess delayed the recovery of one case, and the status of the remaining patient was not determined. On the other hand, of the five who died an autopsy was made in Case XL., but revealed no hydrocephalus, and the records of Cases XXIV., XXXIX., XLV., and LXXXI. are not of enough detail to warrant a diagnosis. Whether the eleven patients with symptoms of hydrocephalus, who were discharged from the hospital later, remained well is not known. We do know that Case V. of our series was discharged on the twenty-third day from the onset of his illness, yet died of hydrocephalus on the sixtieth day.

TREATMENT. Several of the probable cases of hydrocephalus just mentioned which recovered were tapped one or more times, but nothing indicates that this was of especial benefit. No large amount is reported removed and no marked amelioration of symptoms noted. The treatment of the fatal cases was purely supportive and symptomatic, and this holds good for the others. In the literature recently there have been many reports on the efficacy of lumbar puncture in hydrocephalus after meningitis, but a study of these has failed to convince me of its usefulness. And how improvement can take place by this or any other means is very hard to see. The sudden death within twenty-four hours in Case V., following unusual exertion after a long period of convalescence, shows how careful one must be in the care even at a time when a cure appears established.

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[1] Cyclopædia of the Practice of Medicine, vol. ii., p. 728.

[2] Report of the State Board of Health of Massachusetts, p. 129.

# A CRITICAL SUMMARY OF THE LITERATURE

ON THE

## DIAGNOSTIC AND THERAPEUTIC VALUE OF LUMBAR PUNCTURE.

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SINCE H. Quincke's[1] epoch-making contribution in 1891, which showed appreciation of a hitherto unused anatomical fact and opened up a wide realm for investigation, there has accumulated a vast amount of literature dealing with removal of cerebro-spinal fluid through a needle introduced into the subarachnoid space between the transverse processes of the lumbar vertebræ, or, as it is briefly expressed, lumbar puncture. So extensive is the literature that only those articles will be here referred to which bring out the different points of the subject, the conclusions being based on a general review of all accessible articles.

R. Neurath[2] has given a very exhaustive review of the subject up to 1898; and while this article will, to a certain extent, be supplementary to Neurath's, it will, of necessity, touch on many of the articles which appeared prior to two years ago.

Quincke first used the operation for the relief of excessive intracranial pressure in cases of chronic hydrocephalus, to supplant the more formal operation of tapping the ventricles, which procedure necessarily often caused trauma to a limited region of brain-substance. From this as a start, the field of lumbar puncture has widened, so that it may now be said to have therapeutic and diagnostic indications. The therapeutic indications are: (*a*) to relieve excessive pressure by the cerebro-spinal fluid on the brain and cord; (*b*) to remove active deleterious bodies which may be present in the cerebro-spinal fluid.

The causes of increased tension in the cerebro-spinal fluid are: all

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[1] Verhandlung. des Congresses f. innere Medicin, 1891, x.

[2] Centralblatt f. die Grenzgebieten der Medizin und Chirurgie, vol. i., 1897-98.